Epitomes

Important Advances in Clinical Medicine

Occupational Medicine

The Scientific Board of the California Medical Association presents the following inventory of items of progress in occupational medicine. Each item, in the judgment of a panel of knowledgeable physicians, has recently become reasonably firmly established, both as to scientific fact and important clinical significance. The items are presented in simple epitome, and an authoritative reference, both to the item itself and to the subject as a whole, is generally given for those who may be unfamiliar with a particular item. The purpose is to assist busy practitioners, students, research workers, or scholars to stay abreast of these items of progress in occupational medicine that have recently achieved a substantial degree of authoritative acceptance, whether in their own field of special interest or another.

The items of progress listed below were selected by the Advisory Panel to the Section on Occupational Medicine of the California Medical Association and the summaries were prepared under its direction.

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Carpal Tunnel Syndrome—A Cumulative Trauma Disorder

REPEATED MECHANICAL STRESS can result in low-level injuries or "cumulative trauma," and there is increasing evidence that certain repetitive or forceful motions of the hands and wrists can contribute to the development of the carpal tunnel syndrome. Postulated mechanisms by which cumulative trauma might cause the disorder include one or more of the following: tendinitis, with attendant compression of the median nerve within the carpal tunnel; local ischemia; and injury to the myelin sheath or disruption of axonal flow, or both.

In epidemiologic studies of working populations, a number of mechanical risk factors for the development of the carpal tunnel syndrome have been proposed. A case-control study of the syndrome among production seamsters found an association with using a deviated wrist or pinching hand position, particularly during forceful exertion. In another casecontrol study examining aircraft assembly workers, the use of vibratory hand tools and, to a lesser degree, repetitive wrist motions were associated with the carpal tunnel syndrome. In a recent cross-sectional study of 652 industrial workers, the prevalence of the syndrome was examined in relation to the postural and force requirements observed for hand and wrist activities at each job. The disorder was clinically diagnosed more than 15 times more frequently among workers classified as having "high force-high repetitive" jobs than among those with "low force-low repetitive" jobs. A high degree of repetitiveness was found to be more strongly associated with the carpal tunnel syndrome than was a high degree of force. The carpal tunnel syndrome should be recognized as a possible cumulative trauma disorder when a patient's occupational or nonoccupational activities include repetitive or forceful hand-gripping or wrist-deviating motions or the use of vibrating tools.

There is evidence supporting the efficacy of surgical and nonsurgical therapy in the care of patients with the carpal tunnel syndrome. In cases due to work-related factors, however, no studies have evaluated whether surgical or nonsurgical therapy will better prevent recurrence if patients return to their previous occupations. For these reasons, when an occupational cause for the syndrome is considered, treatment should include the avoidance of repetitive hand stress and the

use of wrist splinting and anti-inflammatory medications as a first line of therapy. The use of pyridoxine in the treatment of the carpal tunnel syndrome has been suggested, but there are little data regarding its efficacy in work-related cases. If conservative measures fail, surgical intervention should be considered.

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REFERENCES

Armstrong TJ, Chaffin DB: Carpal tunnel syndrome and selected personal attributes. J Occup Med 1979 Jul: 21:481-486

Dawson DM, Hallett M, Millender LH: Carpal tunnel syndrome, chap 2, Entrapment Neuropathies. Boston, Little, Brown, 1983, pp 5-58

Silverstein BA, Fine LJ, Armstrong TJ: Occupational factors and carpal tunnel syndrome. Am J Ind Med 1987; 11:343-358

Expanding Awareness of the Hazards of Vinyl Chloride

VINYL CHLORIDE MONOMER is a gas that can be converted into polyvinyl chloride resin, which, with the addition of various stabilizers and plasticizers, may be converted into pellets, powders, pastes, or film, the sum of which will constitute 35% to 40% of plastic products made. Depending on the manufacturing process, the polyvinyl chloride resin will contain a varying proportion of residual vinyl chloride monomer that will vaporize subsequently, although this proportion has been greatly diminished during the past decade. If polyvinyl chloride burns, a small but consistent release of vinyl chloride occurs, although the major toxic combustion products are hydrochloric acid and carbon monoxide.

Vinyl chloride has long been known to be hepatotoxic and a central nervous system depressant. For more than two decades we have known that it causes the development of acroosteolysis or of Raynaud's phenomenon in certain exposed workers, and the identification of a characteristic vascular abnormality has explained the probable pathogenesis of these conditions. Vinyl chloride has also been reported to be the cause of "vinyl chloride disease," a syndrome resembling one or more of the collagen diseases, such as scleroderma, rheumatoid arthritis, systemic lupus erythematosus, or the mixed collagen syndrome.

The carcinogenicity of high doses of this gas in rats was first reported in 1970, and human exposure leading to an

unusual type of liver tumor—angiosarcoma—was reported in 1974. The accumulated reports of an increased risk of hepatic angiosarcoma associated with an antecedent exposure to vinyl chloride have achieved almost the same degree of acceptance in current medical wisdom as the association between mesothelioma and asbestos. Just as asbestos causes more cancers than mesothelioma, however, the carcinogenic activity of vinyl chloride is much broader than hepatic angiosarcoma. After absorption, it is metabolically converted to chloroethylene epoxide, which is highly reactive and binds covalently to nucleic acids, increasing the mutation rate in any tissue that can support this metabolic change. On this basis, it is not surprising that a 1981 review of the epidemiologic studies of workers exposed to vinyl chloride has shown a significant excess risk in four of eight studies of biliary and digestive system cancers, in five of eight studies of brain tumors, in three of eight studies of respiratory system cancers, and in one of five studies of lymphatic and hematopoietic system cancers. A more recent 27-year study of exposed workers in Norway reported only a single case of hepatic angiosarcoma but an increased risk of colon cancer, lung cancer, melanoma, and thyroid cancer.

The experimental and epidemiologic evidence for the carcinogenicity of vinyl chloride monomer is solid, but appropriate protective measures continue to be overlooked frequently. Because a large proportion of plastics manufacture takes place in plants too small to retain their own medical staff, it follows that many, if not most, cases of cancer associated vith this gas will first come to the attention of nonoccupational-medicine physicians who need to be aware of the growing data base linking exposure to vinyl chloride and an expanding array of cancers. As was the case for asbestos, such medical recognition is likely to be essential to the achievement of effective exposure control.

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REFERENCES

Heldaas SS, Langård SL, Andersen A: Incidence of cancer among vinyl chloride and polyvinyl chloride workers. Br J Ind Med 1984 Feb; 41:25-30

Infante PF: Observations of the site-specific carcinogenicity of vinyl chloride to humans. Environ Health Perspect 1981 Oct; 41:89-94

Williams GM, Weisburger JH: Chemical carcinogens, ch 5, In Klaasen CD, Amdur MO, Doull J (Eds): Casaret and Doull's Toxicology: The Basic Science of Poisons, 3rd Ed. New York, MacMillan, 1986, p 124

Recognizing the Health Hazards of Ethylene Oxide

ETHYLENE OXIDE (C₂H₄O) is an important industrial chemical used in sterilization and many manufacturing processes. At ambient temperatures, it is a gas. In vivo, it is rapidly distributed throughout the body. Short- and long-term exposures lead to respiratory tract irritation and may lead to central nervous system depression and seizures. At high concentrations, ethylene oxide can induce lethal mutations and cause embryotoxicity in rodents. It is able to alkylate DNA-causing gene mutations leading to sister chromatid-exchange abnormalities and chromosomal damage. Several types of tumors have been described in laboratory animals associated with ethylene oxide exposures.

Five longitudinal epidemiologic studies in occupationally exposed workers in Sweden, the United States, and West Germany strongly support the association of the agent with leukemia. The data cannot be considered conclusive, as the number of workers is relatively small and it is impossible to

exclude completely exposure to other workplace carcinogens. Nevertheless, these epidemiologic studies strongly suggest that ethylene oxide is carcinogenic in humans. Studies of hospital workers with long-term ethylene oxide exposure, when compared with control populations with no significant difference in educational background, suggest that neurologic dysfunction may result from such exposure. These effects may occur at exposure levels that are common in hospital sterilizing procedures. The use of ethylene oxide is primarily limited to the sterilization of medical and food products.

Because the safe level of exposure to this agent is difficult to define, persons working in close proximity to hospital, laboratory, or food sterilizers should be informed as to the known and the uncertain risks. The function of sterilizing equipment should be regularly assessed to ensure there is no human exposure. The rationale for both environmental and medical surveillance needs to be presented to all potentially exposed persons.

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REFERENCES

Estrin WJ, Cavalieri S, Becker CE, et al: Evidence of central nervous system dysfunction in chronic ethylene oxide exposure. Arch Neurol, in press

Hertz-Picciotto I, Neutra RR, Collins JF: Ethylene oxide and leukemia (Letter). JAMA 1987 May 1; 257:2290

Carcinogenicity of Synthetic Mineral Fibers

SYNTHETIC MINERAL FIBERS such as fiberglass and mineral wools have assumed significant industrial importance and currently represent a \$3 billion domestic industry. The durability, strength, and insulating properties of these fibers allow them to serve a wide variety of purposes, including insulation and structural support. Recognized health risks associated with synthetic mineral fibers include respiratory and skin irritation. They have been considered safe from the standpoint of cancer risk, making their use attractive in applications previously limited to asbestos fibers. Recent epidemiologic data suggest, however, that synthetic mineral fibers may be associated with increased lung cancer risk.

Concern over possible carcinogenic effects was raised in the early 1970s when it was shown that mesotheliomas could be caused in animals by instilling vitreous fibers into the pleural space. Fibers that were long, thin, and durable showed a carcinogenic potential equivalent to asbestos. Inhalation studies in animals, however, which probably represent a more appropriate model of human exposure, did not show these fibers to be carcinogenic or significantly fibrogenic.

Epidemiologic studies in human populations have also raised important questions regarding the health risks associated with synthetic mineral fibers. An increased prevalence of minimal interstitial changes has been found in chest radiographs of insulation plant workers exposed to these fibers. A large cooperative European study of more than 20,000 workers employed in the industry since the late 1930s showed an excess of lung cancers. Subjects whose first exposure occurred more than 20 years before the diagnosis of cancer and who began work in the early technologic phase of the industry's development were most strongly affected and had about a twofold increased lung cancer mortality. Similar results were reported among American workers in the industry. In comparison, lung cancer deaths among asbestos workers are increased fivefold in nonsmokers and 50-fold in smokers. Insufficient data are available to address the role of smoking